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A Unique Problem of Muscle Adaptation From Weightlessness - The Deceleration Deficiency

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Skeletal muscles perform multiple functions in man including mechanical, thermogenic, protein storage, and even cosmetic. It is generally the mechanical function that has priority in the discussion of muscle physiology, since all interactions of man with his environment require coordinated muscle actions and without which we could not even breathe. From the viewpoint of the space traveler, it is most likely that the mechanical function of muscles needs to be given the most consideration especially considering the known effects of weightlessness on muscle atrophy. The mechanical actions of muscle can be separated into three categories: motor, spring, and shock absorption. It is primarily the latter two that are discussed in this presentation since they both concern the action of muscles in opposing external loads or eccentric loading. The use of eccentric loading (muscle lengthening while maintaining tension) as a diagnostic aid and a therapeutic intervention in musculoskeletal disorders has been of recent interest and is discussed as it relates to space travel.

A few years ago, while sitting on a panel evaluating some research sponsored by NASA (ref. 1), I became aware that the only established benefit of an exercise program for astronauts was treadmill running with a bungee cord simulating a one-g environment. Even 2 hours of bicycle ergometry did not have the positive effect of a treadmill run. Was there a difference in the type of exercise that could account for this differential response? It was obvious that certain muscle groups, particularly the extensors of the lower limb, are normally involved in deceleration of the body. Dr. Cavanagh has reported that a basketball player lands with approximately 9 times his body weight with no difficulty. Yet, this force must be absorbed primarily by the muscles of the lower limbs. Even during normal locomotion, a sizable force must be decelerated with each heel strike; descending stairs requires even more force absorption. Thus, deceleration and muscle-lengthening activities are

important physiological events when gravitational effects are operational - more so if emergency egress requires the individual to drop from any significant height out of the space vehicle in a gravity-influenced environment.

From a physiological standpoint, two unique events occur when a muscle is stretched while maintaining tension: (1) the tension per myofiber is greater than that produced even during maximal isometric conditions (refs. 2 and 3) and (2) sensory receptors responding to both length (stretch) and tension are stimulated simultaneously. Since tension is generally accepted as the stimulus for muscle growth (ref. 4), muscles undergoing eccentric contractions and, thus, experiencing higher forces during normal locomotion should be stronger or more susceptible to atrophy when the stimulus is removed. Documentation for both conditions exists (refs. 5 to 7). Likewise, additional sensory input is derived from muscle-lengthening activities (e.g., walking) as compared to muscle-shortening activities (e.g., bicycle exercises). Could it be possible that the decrease in ambulation performance seen in astronauts and cosmonauts upon return to Earth (ref. 6) was due to a combination of muscle atrophy and sensory derivation?

With the advent of machines that can test muscle activities in all modes of dynamic and static muscle function in combination with electromyographic (EMG) data, it should soon be possible to document deficits and plan rehabilitative or preventative exercise programs suitable for in-flight use. One such device, the Kin/Com, has been used by us for approximately 4 years. The machine (a robotic dynamometer) employs standard hydraulic and computer systems, both of which might be expected to be present on an aircraft. The first series of studies on the Kin/Com involved testing subjects without any deficits or symptoms. These led to some surprising observations. Force oscillations were noted to occur in the knee extensor musculature only during

eccentric contractions of maximal effort. Further investigations supported the hypothesis that these force oscillations were induced by actions of sensory receptors because they were of a frequency excessively high for voluntary control and were diminished with cooling (ref. 8) - a known inhibitor of stretch reflexes.

The presence of force oscillations during eccentric exercise resulted in force-velocity relationships (Stauber, unpublished observations) that were not expected from the studies on isolated muscles or calculations from the Hill equation. The extremely high forces predicted from in vitro experiments did not occur - perhaps because of reflex-mediated inhibitions. Deviations from the predicted force-velocity relationships have also been observed to occur during concentric exercises of the quadriceps muscles when performed at very slow speeds (ref. 9). Thus, complex neurologic control mechanisms exist for intact muscles which need to be studied before the function of sensory input and muscle output can be fully understood.

In another series of experiments, a patient population was identified which had a deficit in the eccentric loading capability of their quadriceps muscles that occurred along with their knee pain. Exercises designed to alleviate this deficit also relieved their knee pain (ref. 10). These observations supported the theory that a decelerator deficit can occur and lead to musculoskeletal problems.

What was the mechanism for such a deficit? The accepted rationale for the occurrence of anterior knee pain syndrome was a biomechanical disorder resulting from large patellofemoral reaction forces causing an irritated and inflamed surface to scrape past another. However, in our study, these forces would have been created during the concentric loading where there was not a deficit. Alternatively, this problem might have to do with the control of the muscles during muscle-lengthening activities causing patellar malalignment.

Evidence for a motor control problem was confirmed when an individual without symptoms but having a history of multiple subluxations of one patella was tested with EMG recordings along with the force records. As with the patients in the study (ref. 10), he demonstrated a marked force drop only during eccentric exercises and in the range of motion between 30° and 60° of knee flexion. The EMG recording illustrated that this force deficit was preceded by a silent period in muscle action potentials even though the subject was attempting a maximal effort (ref. 11). Since there was no such absence of EMG's during the concentric exercise, primary

neuromuscular disease can be ruled out. Instead, some type of motor control problem below the level of conscious activation of muscles seemed operative.

There have been reports that this predisposition to subluxing patellae has a genetic component and is often present in members of the same family (ref. 12). Next, we tested three generations of one family because the youngest member had a marked force deficit and had been the subject for the EMG study mentioned previously. The other members of the family were asymptomatic at the time of the test, but one member of each generation nevertheless demonstrated some measurable deficit, although the magnitude of the deficit was quite variable. Could it be possible that the problem arises if the individual with such a deficit either becomes fatigued or loses muscle strength because of atrophy? Unfortunately, insufficient evidence is available at this time to answer this question. However, the incidence of patellar subluxations reported by the subjects used for EMG testing always occurred when the subject was most fatigued (i.e., at the end of a day of water skiing, etc.). In addition, Dr. Walsh of the Houghston Clinic reported (personal communication) that anterior knee pain did result in patients who were immobilized for orthopedic problems not related to the knee, as if a loss of a certain amount of protective force capability might place these asymptomatic but predisposed individuals into the symptomatic group. Could this also be a potential problem for space travelers - especially related to their ability to perform an emergency egress where a subluxed patella might prevent escape from the area around a disabled spacecraft? This possibility certainly needs further investigation.

In summary, the focus of this report has centered around decelerator problems of the knee, since the lower leg musculature is known to atrophy in response to weightlessness. However, other important decelerator functions are served by the shoulder muscles, in particular the rotator cuff muscles. Problems in these muscles often result in tears and dislocations as seen in baseball pitchers. During this workshop, we have seen photographs of astronauts holding satellites. Would fatigue in their shoulder muscles have the potential for shoulder subluxations and how could these be prevented?

Obviously, many questions have been raised that need further documentation. Since this workshop has been designed around providing information as to the existence of problems that might need exercise prescriptions as well as indicating which devices might be used to measure and exercise

space travelers in an attempt to mitigate potential problems, it is noteworthy that at least one device currently exists that can measure concentric and eccentric muscle loading including a submaximal simulated free weight exercise (i.e., force-controlled) and simultaneously record integrated EMG analysis appropriate for assessment of all muscle functional activities. Studies should be undertaken to provide information as to the performance of maximal and submaximal exercise in space travelers to define potential problems and provide rationale for prevention.

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